NATIONAL OAK WILT SYMPOSIUM

JUNE 4-7, 2007 • AUSTIN, TEXAS

The Proceedings of the 2nd National Oak Wilt Symposium

Edited by:

Ronald F. Billings
David N. Appel

Sponsored by

International Society of Arboriculture – Texas Chapter

Cooperators

Texas Forest Service
Texas AgriLife Extension Service
The Nature Conservancy of Texas
Lady Bird Johnson Wildflower Center
USDA Forest Service, Forest Health Protection

2009

EPIDEMIOLOGY AND OCCURRENCE OF OAK WILT IN MIDWESTERN, MIDDLE, AND SOUTH ATLANTIC STATES

Jennifer Juzwik
USDA Forest Service
Northern Research Station
1561 Lindig Avenue
St. Paul, MN 55108
Email: jjuzwik@fs.fed.us

ABSTRACT

In Midwestern, Middle, and South Atlantic states, the oak wilt fungus (*Ceratocystis fagacearum*) is transmitted from diseased to healthy oaks below ground via root grafts and above ground via insect vectors. Recent studies have identified insect species in the family Nitidulidae that likely account for the majority of above-ground transmission during spring in several Midwestern states based on frequencies of fungus-contaminated beetles dispersing in oak stands and visiting fresh wounds. Other investigations have utilized quantitative and spatial data to predict root-graft spread in red oak stands. Although the disease is widely distributed in the regions, disease severity ranges from low to high among the regions and within states of the Midwestern region. Knowledge of spread frequencies and relationships between disease spread/severity and various physiographic factors is important in the development of tools for effective disease management.

Key words: *Ceratocystis fagacearum*, disease spread, Nitidulidae

New oak wilt infection centers (= foci) are the result of above-ground transmission of the pathogen (*Ceratocystis fagacearum* (Bretz) Hunt) by animal vectors, primarily insects. Outward expansion of foci from the initial infection(s) occur below ground when fungal propagules move through vascular root connections between a diseased and a nearby healthy oak. A basic understanding of these general means of oak wilt spread was developed at least 40 years ago and was a result of intense research activity that occurred in the 20 years following the recognition and description of *C. fagacearum* as the causal organism (Henry et al. 1944). An excellent review of oak wilt transmission was published in 1980 (Gibbs and French 1980). Modifications of this understanding were subsequently made in response to the first recognition of oak wilt in Texas and the specifics of pathogen spread that were elucidated thereafter (Appel 1995). Refinements and additional details of spread in the Midwestern States, Middle, and South Atlantic states have also occurred during the past three decades.

Documentation of disease foci occurrence across landscapes is available in the older literature, but factors influencing the distribution of *C. fagacearum* are complex and have been poorly understood (MacDonald 1995). Landscape-level epidemiological models and emerging spatial tools are providing increased understanding of various factors and features correlated with disease patterns across landscapes (e.g., Bowen and Merrill 1982, Menges and Loucks 1984, Appel and Camilli 2005). Quantitative data available in published literature can be valuable in such investigations; disease prediction efforts may also benefit from such information. New or refined tools for predicting, preventing, monitoring, or managing spread of the oak wilt fungus in forest landscapes arise from quantitative and spatial studies of the disease occurrence and spread.

The epidemiology of oak wilt outside Texas was most recently reviewed by Prey and Kuntz (1995). In this paper, aspects of the epidemiology and occurrence of oak wilt within and among states of the Midwestern, Middle Atlantic, and South Atlantic regions of the U.S. are considered. Particular attention is paid to results of recent studies (< 30 years). The frequencies of above- and below-ground spread occurrence, factors influencing spread, and relationship of one spread type to the other are discussed within the context of disease incidence and severity observed in these landscapes. Lastly, applications of this knowledge for effective oak wilt management in these regions are also briefly discussed.

EPIDEMIOLOGY AND DISEASE OCCURRENCE

In the Midwestern and Middle and South Atlantic States, *Ceratocystis fagacearum* is transmitted from diseased to healthy oaks either above ground by insects or below ground through functional root grafts. The pathogen's spread through root grafts results in the progressive enlargement of existing oak wilt foci, whereas insect vectors are responsible for the introduction of the pathogen to previously-unaffected forest patches or non-systematically to healthy trees within contiguous forests where the disease is already established.

Initiation of New Disease Foci

Squirrels, birds, and several insect families have been implicated as above-ground vectors of the pathogen, but little published data exists to support these assertions for the first two groups. Overall, insects are considered to be responsible for the vast majority of above-ground spread. Sap beetles (Coleoptera: Nitidulidae) and oak bark beetles (Coleoptera: Curculionidae: Scolytinae) are commonly cited as the main vector groups while ambrosia beetles (Coleoptera: Curculionidae: Scolytinae), certain buprestids (Coleoptera: Buprestidae) and cerambycids (Coleoptera: Cerambycidae) may be occasional vectors (Merrill and French 1995). Aspects of the two main vector groups are presented here.

Sap Beetles. The sequential conditions needed for successful transmission by sap beetles include: a) the availability of viable inoculum (i.e., oak wilt fungal mats), b) inoculum acquisition by vector species, c) dispersal of contaminated insects, d) attraction of pathogen-contaminated insects to fresh, xylem-penetrating wounds on healthy oaks, and e) receptivity of fresh wounds to infection. The factors influencing several of these conditions have been previously reviewed (Gibbs and French 1980). In general, the highest frequency of sap beetle-mediated transmission occurs during spring months when peaks in mat abundance, contaminated insect density, and host susceptibility coincide. The starting and ending dates of the critical spring period (i.e., for sap beetle transmission) do change with increasing latitude. For example, the month with the highest risk of transmission in central Missouri is April, but in Minnesota it is May based on frequencies of pathogen-contaminated sap beetles captured in fresh wounds on healthy oaks in each state (Juzwik, Skalbeck and Neuman 2004, Hayslett, Juzwik and Moltzan 2008).

Very low to no transmission occurs in November through February when no or only greatly deteriorated mats are present, vector species are in over-wintering locations, and pathogen infection of wounds rarely if ever occurs due to low ambient temperatures. Although late-summer and fall mat production is common and often comparable to levels during spring in Minnesota (Juzwik 1983), the contaminated insect densities of two dispersing sap beetle vector species were low between August and mid-October (Ambourn, Juzwik and Moon 2005) and fresh wounds were not attractive to such pathogen-contaminated species during this same time period (Juzwik et al. 2006). Climatic conditions affect the onset, duration, and abundance of mat production during spring in Minnesota (Juzwik 1983). Average minimum February temperature and total spring precipitation were correlated with number of mats per tree based on mixed effects modeling results (McRoberts and Holdaway, unpublished report on file with USDA Forest Service, St. Paul, MN).

Oak species composition and species densities in oak forests affects the frequency and abundance of inoculum available for vector spread and, theoretically, the frequencies of new center establishment in the landscape. In general, oak wilt fungal mats are commonly formed on red oak species and less frequently to rarely on white oak species. Mats have been commonly observed on the predominant red oak species in the three regions. Contrary to earlier reports (e.g., Berry and Bretz 1966), mats have been found to commonly occur on recently wilted red oaks in Missouri within the past decade (Juzwik and Moltzan, personal observation) and sap beetles commonly inhabit the mats. Because of their propensity for inoculum production, red oaks are a significant factor in *C. fagacearum* spread and disease intensification in forest landscapes of the three regions.

Mat formation has also been reported on inoculated bur oak (*Q. macrocarpa*), a white oak species, in Iowa and Wisconsin (Engelhard 1955, Nair and Kuntz 1963) and, anecdotally, on bur oak in natural landscapes in Minnesota. Furthermore, *C. fagacearum* infected bur oaks may produce new mats in successive years on trees with recurrent wilt symptoms (Nair 1964). Pathogen contaminated sap beetles were associated with the small sporulating mats found on this species (Nair and Kuntz 1963). Mats apparently infrequently to rarely occur on *Q. alba*, another white oak species (Cones 1967). Thus, bur and white oak would appear to play a role, albeit likely minor, in the overland spread of *C. fagacearum* in the three regions.

Oak Bark Beetles. Sequential events leading to successful transmission by oak bark beetles (*Pseudopityophthorus minutissimus* and *P. pruinosus*) include: a) reproductive colonization of recently-wilted oaks, b) acquisition of viable pathogen propagules by teneral adults prior to emergence from colonized, diseased oaks, c) dispersal of contaminated insects, and d) inoculation of healthy oaks during maturation feeding in the crowns of healthy oaks (Ambourn, Juzwik and Eggers 2006). Wounds created by the beetles during feeding are considered suitable infection courts for the pathogen. The frequencies of reproductive colonization by *Pseudopityopthorus* spp. in oaks and dispersion of pathogen-contaminated beetles apparently differ greatly among, and even within parts of, the Midwestern, Middle Atlantic, and South Atlantic states (True et al. 1960, Berry and Bretz 1966, Rexrode 1969, Ambourn, Juzwik and Eggers 2006). The highest frequency of oak bark beetle transmission likely occurs during mid- to late spring. The frequencies of pathogen-contaminated *P. minutissimus* dispersing in oak wilt centers in east central Minnesota were 4 to 13 per thousand in May and June (Ambourn, Juzwik and Eggars 2006).

Predominant Insect Vectors. Historically, sap beetles have been considered the primary vectors in six of the seven states in the Midwestern region. Oak bark beetles were cited as the main vectors in Missouri (Rexrode and Jones 1970); however, recent evidence supports the importance of sap beetles in transmission in the state (Hayslett, Juzwik and Moltzan 2008). Sap beetles as well as oak bark beetles and ambrosia beetles have all been considered vectors in the Middle and South Atlantic states (Merrill and French 1995). Sap beetle species are more efficient vectors than oak bark beetles in Minnesota (Ambourn, Juzwik and Eggers 2006).

In separate Illinois and Minnesota studies, Menges and Loucks (1984) and Shelstad et al. (1991) found higher efficiencies of vector spread over short distances and that longer distance spread occurrences are highly stochastic. In Minnesota, contaminated insect densities of the dispersing sap beetle *Colopterus truncatus* were higher when populations were sampled in active disease centers compared to those in oak wilt–free stands (Ambourn, Juzwik and Moon 2005).

Rates of New Foci Occurrence. Landscape-level estimates of the frequencies of new oak wilt centers established via overland spread are available for four Midwestern states. Rates range from a high of 0.42 new centers/ha/yr for Minnesota and Wisconsin (Anderson and Anderson 1963), to much lower in Missouri (< 0.07 new foci/ha/yr) (Jones and Bretz 1958), and lowest in Illinois (≤ 0.006 new foci/ha/yr) (Menges 1978). In comparison, the frequencies of new center occurrence in Pennsylvania and West Virginia are one-tenth to one-hundredth of the lowest rates for the Midwestern states (Merrill 1967). The rates for North Carolina and Tennessee were even lower (< 0.0006 new foci/ha/yr) than for Pennsylvania and West Virginia (Boyce 1959).

Expansion of Disease Foci

Root Grafting and Pathogen Spread via Grafts. Root grafts are known to occur in numerous oak species (Graham and Bormann 1966). When the roots of trees in close proximity graft together and form a functional union, the biological processes of one tree are strongly influenced by those of the connected tree(s) (Epstein 1978).

Self-, intra-specific-, and inter-specific root grafting occur in oaks. Self-grafting is common in red oaks and may facilitate movement of the fungus among the major roots without first passing through the root collar. In a Minnesota study, *C. fagacearum* was isolated from 14 of 62 self grafts assayed from 12 diseased northern pin oaks (Blaedow and Juzwik 2007). Frequencies of intra-specific grafting occurrence vary by species, site, and geographic region. The highest frequencies (over 70%) of such grafting have been reported for *Q. ellipsoidalis* and the lowest for *Q. macrocarpa* (6%), both in central Wisconsin (Parmeter, Kuntz and Riker 1956). Frequencies of inter-specific grafting are generally lower than intra-specific. Such grafting has been reported between species within the red oak group and between species of the red and white oak groups. The highest inter-specific grafting frequencies (43%) reported in the literature occurred between *Q. velutina* and *Q. alba* in North Carolina (Boyce 1959). Grafting between *Q. macrocarpa* and *Q. ellisoidalis* is not uncommon in Minnesota (Juzwik, personal observation).

Other factors influencing frequencies of root grafting, and hence of *C. fagacearum* spread, are basal area (combined measure of tree density and tree diameters), soil depth, soil texture, and occurrence of non-oak species. The percent oak mortality attributable to root-graft transmission and average disease center size increase with increasing percent red oak composition in Midwestern forests (Menges and Loucks 1984). Root grafting frequency was higher in shallower and/or restricted soils in West Virginia (e.g., True and Gillespie 1961, Gillespie and True 1959). Frequencies of root graft spread also increase from heavier textured soils (silt loam) to light textured soils (e.g., sands) (Menges 1978, Prey and Kuntz 1995). The occurrence of non-oak species in affected stands can either reduce the incidence of root graft spread when inter-mixed among the oaks or stop the below-ground spread when the type changes within the stand.

<u>Rates of Disease Foci Expansion</u>. In Midwestern states, the average radial expansion of oak wilt foci ranges from 1.9 to 7.6 m/yr with the highest rates occurring on deep sand soils of the Anoka Sand Plains, Minnesota, and up to 12 m/yr on sandy soils in the Upper Peninsula of Michigan (Bruhn and Heyd 1991). The oak wilt-associated mortality in the latter type sites average 8 to 11 red oaks/ha/yr. Bur oaks die at a much lower rate (≤ 1/ha/yr) (French and Bergdahl 1973). Radial expansion rates in distance measurements for the Middle and Atlantic states are not reported in the published literature. However, mortality rates attributed to root-graft spread are available. In Pennsylvania, mortality rates varied from 1 to 3 oaks/center/yr (Jefferey and Tressler 1969, Jones 1971) while the lowest rates, 0.19 to 0.39 oaks/center/yr, have been reported for West Virginia (Jones 1971, Mielke, Hayes and Rexrode 1983).

Frequencies of Spread Type in Relation to Disease Occurrence

Low frequency to rare occurrence of *Ceratocystis fagacearum* spread across larger distances (e.g, > 300 m), especially from an affected forest patch to a disjunct, wilt-free patch obviously constrains the frequency of within patch spread and development of oak wilt across the landscape (Fig. 1, as modified from Menges and Loucks 1984). If the long-distance event frequency were to increase (e.g., through increased transport frequency of inoculum-laden logs to an area), then the frequencies of within-patch vector spread and root-graft transmission would correspondingly increase, especially if species composition and density, landform, and soils were conducive to spread. Similarly, the frequency of within-patch vector spread which is largely dependent on red oak species abundance directly affects the incidence of new foci within a forest patch and the opportunity for increased root-graft spread, such as occurs in several Midwestern states (Fig. 1A). However, when root-graft frequency is lower, the temporal and numeric incidence of mat-bearing oaks should logically be lower and the probability for both within and among patch vector spread correspondingly lowered as occurs in the Middle and South Atlantic states and southern portions of the Midwestern states (Fig. 1B).

A high red oak component is critical to sustaining an epidemic and thus of increasing the spread frequency. Using the ratio of red oak to white oak volume for a state's oak forest resources (derived from Smith et al. 2004), the calculated ratios for states sustaining oak wilt epidemics ranged from 1.32 to 2.58 for Minnesota, Michigan, and Wisconsin. In contrast, the ratios for Middle and South Atlantic states are < 1.0, except for Pennsylvania with a ratio of 1.27 and < 1.0 in Iowa and Missouri in the Midwest region.

Oak Wilt Occurrence and Other Factors

Topographic position is correlated with oak wilt occurrence. Furthermore, landform and soils are important factors in explaining oak wilt incidence and severity. In areas of Pennsylvania, Wisconsin, and West Virginia with obvious topographic relief, oak wilt was reported to be common on upper slopes and ridge tops (Anderson and Anderson 1963, Cones and True 1967, Bowen and Merrill 1982). Oak wilt is also very common in areas of low topographic relief in southeastern Iowa and different parts of Michigan, Minnesota, and Wisconsin.

In general, oak wilt is most severe on dry and dry-mesic sites that are also considered to be low in productivity in the three regions addressed here. Oaks are particularly well-suited for successfully invading disturbed sites characterized by these moisture regimes and compete particularly well for stand space and canopy position against other woody species on dry sites (Johnson, Shifley and Rogers 2003). Such sites are also described as being "poor quality" or characterized by low site index. For oaks, in general, poor sites have site indices of < 50, medium sites have indices between 50 and 65 while good sites are defined by indices of > 65. A much higher percentage (53%) of oak wilt foci occurred on poor sites in Hampshire Co., WV, than would be expected if randomly distributed (Cones and True 1967). In mixed oak stands of the Sinnissippi Forest, Illinois, oak wilt mortality (vector and root-graft attributed) was significantly higher on poor sites than on medium or good sites for mixed oak and all composition of oak forests (Menges 1978).

Conclusions

Although oak wilt is widely distributed in the Midwestern, Middle and South Atlantic states (http://www.na.fs.fed.us/fhp/ow/maps/ow_dist_fs.shtm, last accessed May 17, 2007), disease severity when measured by numbers of oaks killed per hectare, area affected by oak wilt, and/or number of disease foci per hectare differs greatly between the regions and within the Midwestern states. For example, oak wilt has been reported in all but 3 of 55 counties in West Virginia, disease incidence is sporadic and severity (i.e., oak mortality per disease center) is low. In contrast, oak wilt has been reported in only 25 of over 65 counties with significant amounts of oak forests in Minnesota.

In general, disease severity is very high on the deep sand soils north of Minneapolis and St. Paul and east of Rochester, Minnesota (Albers 2001). In Missouri, oak wilt is widely distributed throughout the Ozarks and central Missouri, although relatively few trees succumb to the disease in each disease center. However, oak wilt severity is more obvious in urban and community forests of the St. Louis and Kansas City metropolitan areas compared to the Ozarks (Bruce Moltzan, personal communication, Missouri Department of Conservation, May 21, 2007). Differences in oak wilt severity can often be explained by physiographic factors and features such as those previously discussed.

IMPLICATIONS FOR DISEASE MANAGEMENT

Knowledge of spread frequencies and significant factors influencing local and higher spatial level spread can, and have been, used to develop tools and strategies to manage oak wilt. Several examples of how such knowledge has been applied to oak wilt management at different spatial scales are discussed by spread type.

Managing Above-ground Spread

The frequencies of short- versus long-distance transmission of *C. fagacearum* by insects support an emphasis on removal of potential spore mat-bearing trees at the site and local control level. Effective use of mechanical disruption to stop transmission via root grafts can be negated when measures to prevent insect transmission are not incorporated, i.e., insect vectors may move the fungus over barrier lines. The impact of infrequent, longer distance (> 300 m) transmission events by insects at the landscape level is also significant in terms of oak loss (Shelstad et al. 1991). Human-transport-mediated, insect transmission of the pathogen through intrastate and interstate movement of firewood and logs from recently-felled, diseased trees is an infrequent event but one that can also lead to significant future losses. Such knowledge is considered when natural resource managers develop strategies to prevent the expansion of the oak wilt range in their states and beyond.

Knowledge of the frequencies of pathogen-contaminated beetles visiting wounds and dispersing in oak stands was utilized in conducting an analysis of the relative probability of disease spread into unaffected oak forests undergoing timber stand improvement or harvest activities (Juzwik, Cummings-Carlson and Scanlon 2008). Statewide guidelines for preventing or reducing oak wilt spread during harvesting activities in oak timberland were based on the risk analysis (http://www.dnr.state.wi.us/org/land/forestry/fh/oakWilt/guidelines.asp, last accessed May 20, 2007).

Managing Below-ground Spread

Statistical analyses and modeling efforts defined the relationship between oak tree diameter and inter-tree distance to the probability of below-ground pathogen spread in Wisconsin (Menges 1978, Menges and Loucks 1984). Later research built on this knowledge developed a model to describe the probability of root graft transmission within one year at high confidence levels based on diameters of source and target trees and the distances between them on loamy sand and sand soils (Bruhn et al. 1991). This model was then used to generate a table to guide foresters in root-graft barrier line placement in Wisconsin (Carlson and Martin 2005).

Predicting General Disease Spread and Severity

Recent efforts to develop risk maps for major insect and disease organisms threatening U.S. forests included an analysis of the potential for oak wilt to cause significant oak mortality within a 15-year period (http://www.fs.fed.us/foresthealth/technology/nidrm.shtml, last accessed May 20, 2007). Both scientific- and experienced-based knowledge were used to develop criteria to rank oak forest susceptibility and/or vulnerability to oak wilt occurrence and severity. The criteria that were variously weighted included proximity to existing oak wilt foci, change in human population, road density, oak stocking level, average number of annual storm events, and stand size.

SUMMARY

Oak wilt continues to cause significant losses of oaks in Midwestern oak forests. The disease is widely distributed in the Midwestern, Middle, and South Atlantic states, but disease severity ranges from low to high among the regions and within states of the Midwestern region. Documentation of spread frequencies and elucidation of factors and physiographic features correlated with low to high disease severity in the landscape have proven useful in developing tools or strategies to prevent, detect, and manage the disease in rural and community forests.

ACKNOWLEDGEMENTS

The author thanks Joe O’Brien and Ryan Blaedow for scientific review of the manuscript.

LITERATURE CITED

Albers, J. 2001. Controlling oak wilt, Pp. 1-7. *In*: Forest Insect & Disease. Newsletter, St. Paul, MN: Minnesota Department of Natural Resources (http://www.dnr.state.mn.us/fid/july01/feature.html).

Ambourn, A., J. Juzwik, and J.E. Eggers. 2006. Flight periodicities, phoresy rates, and levels of *Pseudopityophthorus minutissimus* branch colonization in oak wilt centers. Forest Science 52: 243-250.

Ambourn, A.K., J. Juzwik, and R.D. Moon. 2005. Seasonal dispersal of the oak wilt fungus by *Colopterus truncatus* and *Carpophilus sayi* in Minnesota. Plant Disease Reporter 89: 1067-1076.

Anderson, G.W., and R.L. Anderson. 1963. The rate of spread of oak wilt in the Lake States. Journal of Forestry 61: 823-825.

Appel, D.N. 1995. The oak wilt enigma: Perspectives from Texas. Annual Review of Phytopathology 33: 103-118.

Appel, D.N., and K. Camilli. 2005. Spread of *Ceratocystis fagacearum* across the landscape: What do spatial patterns tell us? Phytopathology 95: S127.

Berry, F.H., and T.W. Bretz. 1966. Small oak bark beetle: A potential vector of oak wilt. Plant Disease Reporter 50: 45-49.

Blaedow, R., and J. Juzwik. 2008. Propiconazole movement into the roots of red oaks and *Ceratocystis fagacearum* survival following macroinfusion treatment. Phytopathology 98: S201.

Bowen, K. and W.C. Merrill. 1982. Oak wilt foci related to ridge-bearing aspect in Pennsylvania. Plant Disease 66: 137-139.

Boyce, Jr., J.S. 1957. Oak wilt spread and damage in the Southern Appalachians. Journal of Forestry 55: 499-505.

Bruhn, J., and R. Heyd. 1992. Biology and control of oak wilt in Michigan red oak stands. Northern Journal of Applied Forestry 9: 47-51.

Bruhn, J.N., J.B. Pickens, and D.B. Stanfield. 1991. Probit analysis of oak wilt transmission through root grafts in red oak stands. Forest Science 37: 28-44.

Carlson, J.C., and A.J. Martin. 2005. Oak wilt management – what are the options? University of Wisconsin Extension Publication. G3590. 6 pp.

Cones, W.L. 1967. Oak wilt mats on white oaks in West Virginia. Plant Disease Reporter 51: 430-431.

Cones, W.L. and R.P. True. 1967. Oak wilt incidence related to site productivity in Hampshire County, West Virginia. Phytopathology 57: 645.

Engelhard, A.W. 1955. Occurrence of oak wilt fungus mats and pads on members of the red and white oak groups in Iowa. Plant Disease Reporter 39: 254-255.

Epstein, A.H. 1978. Root graft transmission of tree pathogens. Annual Review of Phytopathololology 16: 181-182.

French, D.W., and D. Bergdahl. 1973. Oak wilt in Minnesota 1972. University of Minnesota Agricultural Experiment Station Miscellaneous Report 118. St. Paul, MN. 8 pp.

Gibbs, J.N., and D.W. French. 1980. The transmission of oak wilt. USDA Forest Service Research Paper NC-185. USDA Forest Service, North Central Forest Experiment Station, St. Paul, MN. 17 pp.

Gillespie, W.H., and R.P. True. 1959. Three factors which influence the local spread of oak wilt in northeastern counties of West Virginia. Plant Disease Reporter 43: 588-593.

Graham, Jr., B.F., and F.H. Bormann. 1966. Natural root grafts. Botanical Review 32: 255-292.

Hayslett, M.C., Juzwik, J. and B. Moltzan. 2008. Three *Colopterus* beetle species carry the oak wilt fungus to fresh wounds on red oaks in Missouri. Plant Disease 92: 270-275.

Henry, B.W., C.S. Moses, C.A. Richards, and A.J. Riker. 1944. Oak wilt, its significance, symptoms and cause. Phytopathology 34: 636-647.

Jefferey, A.R., and R.K. Tressler. 1969. Oak wilt in Pennsylvania: The 1969 program. Pennsylvania Department of Agriculture, Bureau of Plant Industries, Harrisburg, PA. Unnumbered Report. 9 pp.

Johnson, P.S., S.R. Shifley, and R. Rogers. 2002. The Ecology and Silviculture of Oaks. Oxford: CABI Publishing. 503 pp.

Jones, T.W. 1971. An appraisal of oak wilt control programs in Pennsylvania and West Virginia. USDA Forest Service Research Paper NE-204. Northeast Forest Experiment Station, Upper Darby, PA. 15 pp.

Jones, T.W., and T.W. Bretz. 1958. Experimental oak wilt control in Missouri. Missouri Agricultural Experiment Station Research Bulletin 657. 12 pp.

Juzwik, J. 1983. Factors affecting overland transmission of *Ceratocystis fagacearum* in Minnesota. Ph.D. Thesis, University of Minnesota, St. Paul, MN. 96 pp.

Juzwik, J., T.C. Skalbeck, and M.F. Neuman. 2004. Sap beetle species (Coleoptera: Nitidulidae) visiting fresh wounds on healthy oaks during spring in Minnesota. Forest Science 50: 757-764.

Juzwik, J., J.E. Cummings-Carlson, and K. Scanlon. 2008. Risk analysis and guidelines for harvest activities in Wisconsin oak timberlands to minimize oak wilt threat. Forest Encyclopedia Network: Environmental Threats: Case Studies. USDA Forest Service, Asheville, NC. Online pub.(http://www.forestryencyclopedia.net/p/p5/p3389/p3464)

Juzwik, J., M. Hayslett, J.F. Kyhl, and A.K. Ambourn. 2006. Low risk of oak wilt pathogen spread by sap beetles during summer and fall. Phytopathology 96: S57.

MacDonald, W.L. 1995. Oak wilt: an historical perspective, Pp. 7-13. *In*: D.N. Appel and R.F. Billings (eds.), Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium. June 22-25, 1992, Austin, TX. Information Development Inc., Houston, TX.

Menges, E.S. 1978. Patterns of oak mortality in Midwestern oak forests. Proceedings of the Central Hardwood Forestry Conference. 2: 508-528.

Menges, E.S., and O.R. Loucks. 1984. Modeling a disease-caused patch disturbance: oak wilt in the Midwestern United States. Ecology 65: 487-498.

Merrill, W. 1967. The oak wilt epidemics in Pennsylvania and West Virginia: An analysis. Phytopathology 57: 1206-1210.

Merrill, W., and D.W. French. 1995. Insects and the epidemiology of oak wilt, Pp. 29-39. *In*: D.N. Appel and R.F. Billings (eds.), Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium. June 22-25, 1992, Austin, TX. Information Development Inc., Houston, TX.

Mielke, M.E., C. Haynes, and C.E. Rexrode. 1983. Local spread of oak wilt in northeastern West Virginia during 1970-1982. Plant Disease 67: 1222-1223.

Nair, V.M.G. 1964. Pathogenesis of oak wilt in bur oaks. Ph.D Thesis, University of Wisconsin, Madison. 152 pp.

Nair, V.M.G., and J.E. Kuntz. 1963. Mat formation in bur oaks infected with *Ceratocystis fagacearum*. Wisconsin College of Agriculture Forest Research Note 95. 6 pp.

Parmeter, Jr., J.R., J.E. Kuntz, and A.J. Riker. 1956. Oak wilt development in bur oaks. Phytopathology 46: 423-436.

Prey, A., and J.E. Kuntz. 1995. Epidemiology of oak wilt outside of Texas, Pp. 15-20. *In*: D.N. Appel and R.F. Billings (eds.), Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium. June 22-25, 1992, Austin, TX. Information Development Inc., Houston, TX.

Rexrode, C.O. 1969. Population increase of two species of *Pseudopityophthorus* on oak branches. Annals of the Entomological Society of America 62: 448-449.

Rexrode, C.O., and T.W. Jones. 1970. Oak bark beetles – important vectors of oak wilt. Journal of Forestry 68: 294-297.

Shelstad, D., L. Queen, D. French, and D. Fitzpatrick. 1991. Describing the spread of oak wilt using a geographical information system. Journal of Arboriculture 17: 192-199.

Smith, W.B., P.D. Miles, J.S. Vissage, and S.A.Pugh. 2004. Forest resources of the United States, 2002. USDA Forest Service, General Technical Report NC-241. North Central Research Station, St. Paul, MN. 137 pp.

True, R.P., and W.H. Gillespie. 1961. Oak wilt and its control in West Virginia. West Virginia Agricultural Experiment Station Circular 112. 18 pp.

True, R.P., H.L. Barnett, C.K. Dorsey, and J.G. Leach. 1960. Oak wilt in West Virginia. West Virginia Agricultural Experiment Station Bulletin. 448T. 119 pp.

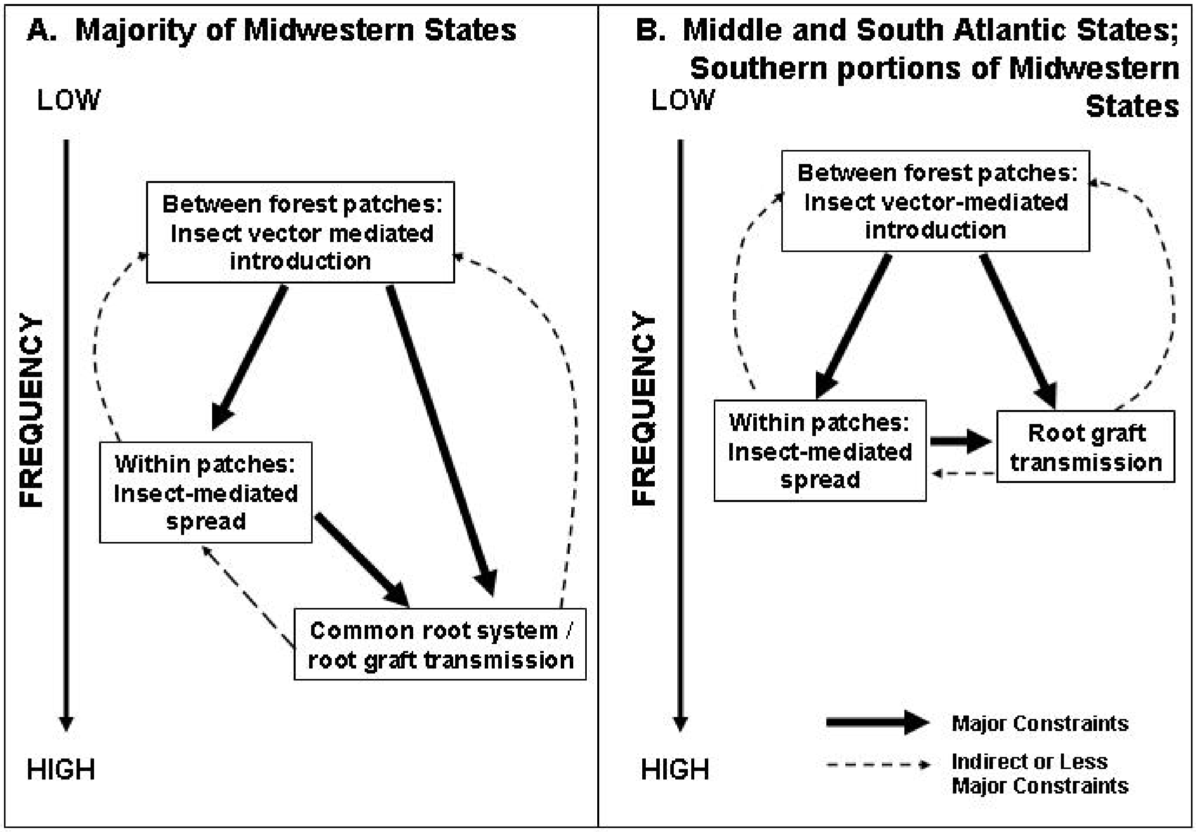

Figure 1. Models of oak wilt occurrence and spread in Midwestern, Middle Atlantic, and South Atlantic states. Figure 1A reproduced with permission from Menges and Loucks, 1984; Figure 1B is a modification of 1A by J. Juzwik.